

Minimal Intervention Dentistry: Systematic Review**Dushyant Pal Singh¹, Saurabh Jain^{2*}, Chetan Sharma³**¹Assistant Professor, Department of Dentistry, Govt. Medical College, Chittorgarh.²Assistant Professor, Department of Dentistry, Govt. Medical College, Dungarpur.³Associate professor, Department of Prosthodontics, RRDCH, Udaipur.

Received: 20-03-2023 / Revised: 11-04-2023 / Accepted: 05-05-2023***Corresponding author: Dr. Saurabh Jain****Conflict of interest: Nil**

Abstract

Introduction: All work in the health field is aimed basically at conservation of the human body and its function. Loss of even a part of a human tooth should be regarded as a serious injury, never to be considered lightly, and the tooth is certainly worthy of the most careful restoration.

Aim and Objectives: To assess of MID is to keep teeth healthy and functional for life. A most important element is achieved through implementing the important strategies for keeping teeth free from carious lesions.

Materials and Methods: Types of studies included in the review Early caries detection and risk assessment, Remineralisation of demineralised enamel and dentine, Optimal caries preventive measures, minimally invasive operative interventions, Repair rather than replacement of restorations.

Results: Fluoride during the remineralisation/demineralisation cycle leads to its incorporation into the crystalline structure of the carbonated hydroxyapatite, which not only decreases crystal solubility, but also increases the precipitation rate of enamel mineral in the presence of calcium and phosphate due to the lower solubility of fluorapatite.¹⁵The fluoride decreases enamel solubility in two ways: (1) the fluoride ion is more stable in the crystal lattice than the hydrogen ion and (2) it interacts with the calcium ions on the crystal surface, interacting closely and binding strongly.

Conclusion: Minimally invasive dentistry requires a change in philosophy in our approach to managing dental caries. Dental caries needs to be viewed as a bacterial disease rather than the end product of that disease—a hole in the tooth.

Keywords: Minimal Intervention Dentistry (MID), Dental caries, GIC.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Preservation of a healthy set of natural teeth for each patient should be the objective of every dentist. All work in the health field is aimed basically at conservation of the human body and its function. Loss of even a part of a human tooth should be regarded as a serious injury, never to be considered lightly, and

the tooth is certainly worthy of the most careful restoration.[1]

Miles Markley, one of several great leaders in preventive dentistry, summarized in this statement the central concept in the modern approach to the dentist's role in the treatment of dental caries: that the loss of even a part of a human tooth should be considered "a

serious injury,” and that dentistry’s goal should be to preserve healthy, natural tooth structure. His words are perhaps even more relevant today than when he wrote them half a century ago, now that we have the scientific understanding and the means to realize his vision.[2]

The “minimally invasive” approach to treating dental caries incorporates the dental science of detecting, diagnosing, intercepting and treating dental caries on the microscopic level. Over time, modern dentistry has evolved to a minimally invasive approach, in which caries is managed as an infectious disease, deferring operative intervention as long as possible. The focus is on maximum conservation of demineralized, nonactivated enamel and dentin.[2] As we already know the aim of MID is to keep teeth healthy and functional for life. A most important element is achieved through implementing the important strategies for keeping teeth free from carious lesions.

Materials and Methods:

These strategies are considered to be:

- a. Early caries detection and risk assessment
- b. Remineralisation of demineralised enamel and dentine
- c. Optimal caries preventive measures
- d. Minimally invasive operative interventions
- e. Repair rather than replacement of restorations.

The oldest method used for detecting carious lesions is tactile examination using mouth mirror and probe. GV Black in 1924 suggested the use of a sharp explorer, based on tug back action for diagnosis of dental caries. However the use of a sharp explorer has been criticized as probing may lead to fracture and cavitation in the incipient lesion. Also there is possibility of transferring cariogenic microorganisms from one site to another, leading to further spread of disease in the same oral

cavity.[3]

Clinical steps:

1. Polymer cup is adapted to primary carious lesion and air is sucked to create a vacuum
2. Ozonated water/ozone gas is delivered at a preset concentration into the cup around the tooth surface.
3. Suction is activated for 10 seconds while cup is still adapted to primary carious lesion to remove residual gas which passes through granular activated carbon filter to remove all traces of ozone.
4. Reductant fluid is pumped for 5 seconds on to the treatment site to start the demineralization process.
5. The patient is instructed to use home care kit.
6. If restoration is required, it is placed after three months.

If restoration is not required, the patient is recalled after three months. Ozone therapy is considered a breakthrough that is expected to be a cornerstone of dental care in the near future.[4]

Detection Devices:

1. Fiberoptic transillumination (FOTI)
2. Quantitative light induced fluorescence
3. Fluorescence camera (vista proof)
4. Laser fluorescence (diagnodent)
5. Electrical conductance measurement

Visual Tactile Methods

Perhaps because of the absence of a properly validated and reliable carious lesion detection device, early enthusiasm dimmed, and the emphasis shifted back to visual-tactile detection methods in the second half of the 1990s. The World Health Organisation (WHO) had propagated its method, which was based on a ‘yes/no’ clearly cavitated dentine lesion, as a reliable data base was required for comparison of decayed, missing and filled (DMF) teeth scores among member countries and because DMF data from decades earlier were available.[23]This

very crude cut-off point, and the fact that caries prevalence and carious lesion development had declined in many industrialized countries, were reasons for epidemiologists to subsequently include the assessment of enamel lesions in caries assessment indices. One such group of epidemiologists developed the International Caries Detection and Assessment System (ICDAS). This two-digit enamel and dentine carious lesion scoring system has recently received much attention. It was developed for use in epidemiological surveys, research, dental education and in practices. The index, when used in epidemiological surveys, has received some criticism[6], could not be applied correctly and was unable to properly allow the reporting of findings[7]. Prior to the launch of ICDAS, Nyvad B, Machiulskiene V and Baelum V (1999)[8] published their 'Nyvad-index', which permits the assessment of enamel and dentine carious lesions as well as the activity/inactivity of enamel carious lesions. The index has been used recently in a number of studies[9,10] and appears to be valid. Monse B, Heinrich-Weltzien R, Benzian H, Holmgren C, van Palenstein Helderman W (2010)[11] introduced the 'Pulpal Involvement Ulcerations Fistula Abscess' (PUFA index) with the intention of alerting the dental / medical / educational communities about the poor state of dentitions of children in the Philippines. A novel visual one-digit caries assessment index was reported recently.[12] It includes non-cavitated and cavitated carious lesions, pulpally involved and abscessed teeth, as well as sealed, restored and lost teeth. In developing the index, experience gained from applying the ICDAS II and PUFA[13] indices in the field were essential. The index is termed Caries Assessment Spectrum and Treatment (CAST). It has been validated for face and content,[14] while construct validity and reliability testing is on-going.

Results

Mechanisms of action of fluoride in enamel:

The presence of fluoride during the remineralisation / demineralisation cycle leads to its incorporation into the crystalline structure of the carbonated hydroxyapatite, which not only decreases crystal solubility, but also increases the precipitation rate of enamel mineral in the presence of calcium and phosphate due to the lower solubility of fluorapatite.[15] The fluoride decreases enamel solubility in two ways: (1) the fluoride ion is more stable in the crystal lattice than the hydrogen ion and (2) it interacts with the calcium ions on the crystal surface, interacting closely and binding strongly.[16]

The effect and penetration of fluoride into the biofilm on the tooth surface is dependent on the type of fluoridated product and the time of exposure. When a clinical biofilm was exposed to 1,000 ppm (0.22%) sodium fluoride solution, exposure of up to 120 seconds increased plaque surface fluoride concentrations only, while 30-minute exposure allowed penetration of more than 1,000 ppm (0.22%) fluoride up to 900 μm into the plaque. The clinical relevance or practicality of a 30-minute exposure is questionable, apart from placement of high concentration (22,600 ppm F⁻; 5% NaF) fluoride varnish. Thus, the efficacy of intermittently applied professional topical gels and foams is questionable, and the use of high concentration fluoridated varnishes should be encouraged, even in children.[17]

Role of calcium and phosphate:

The pre-eminent role of fluoride in preventive dentistry remains valid. However, the effectiveness of fluoride to remineralise enamel and obtain net mineral gain is limited by the bio-availability of calcium and phosphate ions. If the acid challenge to the enamel is extensive, the salivary calcium and phosphate reservoir is

quickly depleted and a net loss of enamel mineral can occur.[18]

The intrinsic sources of calcium and phosphate are saliva, dissolved tooth mineral and to a lesser degree, gingival crevicular fluid. To gain net remineralisation, the action of fluoride is limited by the amount of calcium derived from saliva, without extrinsic bioavailable sources of calcium and phosphate.[19] Increased concentrations of calcium would also increase the retention of fluoride in the plaque biofilm by increasing calcium-bridging.

Therefore, for remineralisation to occur during increased caries risk, an increase in bioavailable calcium and phosphate is fundamental to improving the effectiveness of the agent. Increased calcium and phosphate can be stabilised by macromolecules inherent in the saliva and plaque. However, the concentration of these proteins and peptides is limited. Therefore, a method for improving the effectiveness of calcium and phosphate stabilisation in the oral environment is required.[20]

The assumed relationship between carious lesion development and consumption of fermentable sugars used to be stronger in the past than currently. The extensive exposure to different kinds of fluoride vehicles is considered the main reason for this situation.[21] Diet control, in terms of intake of sugars and other fermentable carbohydrates, is still an important factor in managing carious lesion development. Individuals at high caries risk, and/or those that do not use fluoride agents, will benefit from dietary control measures. The interplay between consumption of cariogenic food, oral hygiene, availability of saliva and fluoride is nicely modeled by Van Loveren C and Duggal MS.[22] They state that as long as saliva and fluoride are available in the mouth in abundance, and if biofilm control is performed properly at the same time, the detrimental effect of

cariogenic food consumption on demineralizing enamel and dentine can be considered low.

Topical Fluorides

The efficacy of topical fluoride in caries prevention depends on a) the concentration of fluoride used, b) the frequency and duration of application, and to a certain extent, c) the specific fluoride compound used. The more concentrated the fluoride and the greater the frequency of application, the greater the caries reduction.

Systemic Fluoride

Fluoride has systemic mechanism of caries inhibition when it is incorporated into the tooth pre-eruptively. Ingested fluoride is absorbed systemically by calcified tissues, including developing enamel. Therefore, a person's teeth may benefit from early-life exposure to systemic fluoride. The mineralization period varies, in terms of age of initiation and duration, across people and among the different teeth for a given person. Also, ingested fluoride can exert a topical mechanism of action when it is redistributed to the oral environment by means of saliva.

Chlorhexidine is available in mouth rinses, gel and varnish. A systematic review was aimed at determining the carious lesion-inhibiting effect of chlorhexidine varnishes on the permanent dentition of children, adolescents and young adults.

Discussion

Minimal Intervention Dentistry aimed to limit unnecessary removal of healthy tooth structure, and repair of defective restorations is one of its strategies. Although the repair of resin composite restorations has been investigated extensively and found successful,[23] dental practitioners do not routinely consider this treatment option in the management of defective restorations. The repair of resin composite restorations is taught in most, but not in all dental

schools in North America, the United Kingdom, Ireland, Germany and Scandinavia.[24] Although considered a long lasting treatment by the schools teaching this practice, a practice-based research study showed that only practitioners who practiced in non-fee service settings, practitioners with fewer years since graduation from dental school, and practitioners who assessed caries risk, chose preventive treatment options more often than replacement when assessing defective restorations.[25] The preference for replacement of restorations may be the result of a complex interplay between the lack of clear standards for replacing restorations and lack of an existing reimbursement for these treatments. That same study reported that general practitioners would most likely intervene surgically in a defective resin composite restoration but not in a defective amalgam restoration.[25]

So far, prospective studies have shown that repaired restorations in permanent teeth have the same or increased longevity as restorations that were replaced completely.[26] Repair treatment remained stable over a 7-year observation period.[26] Additionally, the reason that repaired restorations may even outlast those that were replaced probably relates to the fact that most of the restoration's original form is kept intact, limiting the introduction of new elements that can affect the success of the restoration. When other restoration stress factors are considered, such as stress on the tooth, post-operative sensitivity, and re-exposure of the dentinal tubules with possible pulpal reactions to thermal or mechanical stimulus,[27] damage to the adjacent tooth and the possibility of more complex restorations, it makes perfect sense to pursue the repair of defective restorations as a predictable and conservative approach to preserving tooth structure. A recent overview regarding restoration margins concludes that margin defects, without

visible evidence of soft dentin on the wall or base of the defect, should be monitored, repaired or resealed, in lieu of total restoration replacement.[28]

Besides being a successful treatment, restoration repair is also practical. Defective restorations can be repaired more quickly and with lower operational costs than replacement. Therefore, repaired restorations could present a reduction in patient and/or the third party payers' expenses which would potentially increase the number of individuals who could afford dental care. The cost of care and oral health are severely impacted by the replacement of existing restorations. Examining outcomes of alternative treatment to the replacement of restorations and establishing consistent criteria that will affect general practitioners' treatment decisions is a critical issue that may profoundly change the over-treatment of existing restorations.

Conclusion

Minimally invasive dentistry requires a change in philosophy in our approach to managing dental caries. Dental caries needs to be viewed as a bacterial disease rather than the end product of that disease—a hole in the tooth. Patients should be assessed for their caries risk. The practitioner can then help patients prevent caries or reverse it in its early stage by interrupting the disease process prior to cavitation of the lesion. This requires suppressing the bacteria, limiting the substrate upon which they survive and its duration in the mouth, enhancing the oral environment (increasing saliva and its minerals), and protecting the teeth with fluoride and sealants in addition to the usual oral hygiene methods that we teach our patients. When the caries process cannot be reversed, minimally invasive techniques and materials should be used to conserve sound tooth structure. Air abrasion is one such treatment modality that allows us to minimize loss of sound

tooth structure during caries removal. The changing nature of fissure.

caries with later cavitation and our inability to accurately detect fissure caries with the dental explorer and radiographs can leave us perplexed as to whether to “watch and wait” or invade the fissures. With air abrasion we can remove debris from suspicious fissures and take a look to determine whether caries is present or not. When debris is removed, if caries is not present we need proceed no further. We can then “seal for prevention” and abandon antiquated principles of extension for prevention. Conservative approaches should help patients maintain their dentition for their lifetimes.

References:

1. Markley M. Restorations of silver amalgam. *J Am Dent Assoc.* 1951; 43(2): 133-46.
2. Carol Anne Murdoch-Kinch, Mary Ellen McLean. Minimally invasive dentistry. *J Am Dent Assoc.* 2003; 134:87-95
3. Nikhil Marwah; *Textbook of Pediatric dentistry*; 3rded.
4. Bogra P, Nikhil V, Singh V, Sharma S, Arora V : Ozone therapy for dental caries –A revolutionary treatment for the future. *JIDA* 2003; 74 (1); 41- 45.
5. Pitts N. ICDAS - an international system for caries detection and assessment being developed to facilitate caries epidemiology, research and appropriate clinical management. *Community Dent Health.* 2004; 21:193–198.
6. de Amorim RG, Figueiredo MJ, Leal SC, Mulder J, Frencken JE. Caries experience in a child population in a deprived area of Brazil, using ICDAS II. *Clin Oral Investig.* 2012; 16:513–520
7. Agustsdottir H, Gudmundsdottir H, Eggertsson H, Jonsson SH, Gudlaugsson JO, Saemundsson SR, Eliasson ST, Arnadottir IB, Holbrook WP. Caries prevalence of permanent teeth: a national survey of children in Iceland using ICDAS. *Community Dent Oral Epidemiol.* 2010; 38:299–309.
8. Nyvad B, Machiulskiene V, Baelum V. Reliability of a new caries diagnostic system differentiating between active and inactive caries lesions. *Caries Res.* 1999; 33:252–260.
9. Nyvad B, Machiulskiene V, Fejerskov O, Baelum V. Diagnosing dental caries in populations with different levels of dental fluorosis. *Eur J Oral Sci.* 2009; 117:161–168.
10. Séllos MC, Soviero VM. Reliability of the Nyvad criteria for caries assessment in primary teeth. *Eur J Oral Sci.* 2011; 119:225–231.
11. Monse B, Heinrich-Weltzien R, Benzian H, Holmgren C, van Palensteinhelderman W. PUFA An index of clinical consequences of untreated dental caries. *Community Dent Oral Epidemiol.* 2010; 38:77–82.
12. Frencken JE, de Amorim RG, Faber J, Leal SC. The Caries Assessment Spectrum and Treatment (CAST) index: rational and development. *Int Dent J.* 2011; 61:117–123.
13. Figueiredo MJ, de Amorim RG, Leal SC, Mulder J, Frencken JE. Prevalence and severity of clinical consequences of untreated dentine carious lesions in children from a deprived area of Brazil. *Caries Res.* 2011; 45:435–442.
14. de Souza AL, van der Sanden WJM, Leal SC, Frencken JE. Caries Assessment Spectrum and Treatment (CAST) index: Face and content validation. 2012 Submitted.
15. ten Cate JM. Current concepts on the theories of the mechanism of action of fluoride. *Acta Odontol Scand.* 1999; 57:325–329.
16. deLeeuw NH. Resisting the onset of hydroxyapatite dissolution through the incorporation of fluoride. *J Physic Chem B.* 2004; 108:1809–1811

17. Marinho VC. Evidence-based effectiveness of topical fluorides. *Adv Dent Res*. 2008; 20:3–7.
18. Featherstone JD. The caries balance: contributing factors and early detection. *J Calif Dent Assoc*. 2003; 31:129–133.
19. Yamazaki H, Litman A, Margolis HC. Effect of fluoride on artificial caries lesion progression and repair in human enamel: Regulation of mineral deposition and dissolution under in vivo-like conditions. *Arch Oral Biol*. 2007; 52:110–120.
20. Reynolds EC. Calcium phosphate-based remineralization systems: scientific evidence? *Austr Dent J*. 2008; 53:268–273.
21. Burt BA, Pai S. Sugar consumption and caries risk: A systematic review. *J Den Educ*. 2001; 65:1017–1023.
22. vanLoveren C, Duggal MS. The role of diet in caries prevention. *Int Dent J*. 2001; 51:399–406.
23. Tezvergil A, Lassila LV, Yli-Urpo A, Vallittu PK. Repair bond strength of restorative resin composite applied to fiber-reinforced composite substrate. *Acta Odont Scand*. 2004; 62:51–60.
24. Blum IR, Lynch CD, Wilson NHF. New horizons in minimally invasive dentistry: contemporary teaching of repair versus replacement of defective direct composite restorations in UK and Irish dental schools. *Eur J Dent Educ*. 2012 (in-press)a.
25. Gordan VV, Garvan CW, Richman JS, Fellows JL, Rindal DB, Qvist V, Heft MW, Williams OD, Gilbert GH. for The DPBRN collaborative group. How dentists diagnose and treat defective restorations: evidence from The Dental Practice-based Research Network. *Oper Dent*. 2009; 34:664–673b.
26. Gordan VV, Riley JL III, Garvan CW, Mondragon E, Blaser PK, Mjor IA. 7-Year results of alternative treatments to defective amalgam restorations. *J Amer Dent Assoc*. 2011; 142:842–849.
27. Hirata K, Nakashima M, Sekine I, Mukoyama Y, Kimura K. Dentinal fluid movement associated with loading of restorations. *J Dent Res*. 1991; 70:975–978.
28. Dennison JB, Sarrett DC. Prediction and diagnosis of clinical outcomes affecting restoration margins. *J Oral Rehabil*. 2012; 39:301–318.